Society Reports.

CHICAGO NEUROLOGICAL SOCIETY.

February 23, 1899.

The President, Dr. Richard Dewey, in the chair.

Dr. Sanger Brown read a paper with the title: "Report of a Case of Trigeminal Paralysis." (See p. 554.)

Dr. H. N. Moyer had observed a case very similar to the one reported by Dr. Brown. Twelve years ago he was consulted by a hostler for paresthesia of the face. He said his face felt as if it had been kicked by a horse the day before. Two or three years later he developed anesthesia of the face and loss of taste. The anesthesia finally became complete and sensation to temperature, touch and pain was absolutely gone. Nevertheless, if the skin was moved he knew that he was touched. This seems to show that there is a muscle-sense apart from the pain- and temperature-sense. Later the patient developed eye symptoms, partial ptosis and paresis of ocular muscles, pains developed, the knee-jerk disappeared, and he is now, twelve years after the first observation, in the Kankakee Hospital, a typical case of tabes.

Dr Moyer reported the case of a man who had complete anesthesia of the middle trunk of the fifth nerve with bilateral paresthesia for several years, but in this case there was no disturbance of taste. He had had various ocular palsies, but of late had lost the knee-jerk, had a good deal of swaying when his eyes were closed and lightning pains.

In a third case there was total anesthesia in the middle trunk of the fifth nerve on one side with no impairment of taste, but with ulceration and opacity of the cornea, loss of knee-jerk and swaying when the eyes were closed.

On grouping these three cases, considerable variation in their symptoms will be seen: One had trophic disturbance of the cornea, one had disturbance of the taste-sense, and the others had not. When these cases are taken in connection with Dr. Brown's case it will be seen that as far as the physiological part of the nerve of taste is concerned, these four cases throw very little light upon it, and it must be considered that the sense of taste differs in different individuals. Dr. Brown's case appeared to be one of tabes.

Dr. Brown stated that the knee-jerks were normal and that there was no ataxia.

Dr. Moyer believed that the knee-jerks would disappear later. In the case he had observed there was not a sign of tabes for the first six or eight years the patient was under observation. He considered the trouble to be not in the Gasserian ganglion, but back of it.

In reply to a question by Dr. Gradle, Dr. Moyer stated that tabes was likely to result at any age and was not excluded by advanced age; and in reply to an inquiry by Dr. Dewey, Dr. Moyer said that in the first of his cases a luetic history was doubtful, in the second lues was probable, and in the third nothing could be determined.

Dr. Sydney Kuh asked Dr. Brown whether the tests for the taste-sense were made by him or the patient. Dr. Brown replied that they were made as a rule by the patient, who was cautioned to keep his tongue out of his mouth and hold the substance on his tongue until he tasted it or felt sure that he was not going to taste it. Dr. Brown made some tests in his office by applying solutions on cotton to the tongue, being careful that there should be no opportunity for the solutions to spread to other parts.

Dr. Kuh thought that the cases reported had thrown some light upon the course of the fibers through which the sense of taste is conducted to the brain. In Dr. Brown's case the first and second branches were affected to a greater extent than the third; in two of Dr. Moyer's cases the first and second branches only were affected, and in one case all three branches. These observations are very similar to those published by Schmidt in the *Deutsche Zeitschrift fur Nervenheilkunde*, 1895, and all taken together seem to show that the fibers of the taste sense should be looked for in the third, and not in the second branch of the fifth nerve. The few observations in which the glossopharyngeal nerve was affected at the base of the brain seem to demonstrate that not all the fibers for the sense of taste pass along that nerve, because there is no record of any cases in which there was absolute loss of the sense of taste excepting in the posterior part of the tongue. The age at which tabes appears depends in great measure upon the age at which syphilis is acquired. He had observed one case of a man 51 years old who had had symptoms of tabes for a few months only, but who had acquired syphilis at the age of 36. In another case, the symptoms of tabes developed after the age of 60. One case is recorded in which the first symptoms of tabes occurred at 70, and in this case syphilis was acquired late in life.

Dr. J. J. M. Angear believed that the facial as well as the fifth nerve must be involved in this case. He himself had had paralysis of the facial nerve a few years ago and suffered for a few days from bad taste, although there was no difficulty with the fifth nerve. The origin of the fifth nerve is so extensive that part of it goes to the lower part of the cervical portion of the cord, and a part goes up to the fourth ventricle, the aqueduct of Sylvius, etc. The nuclei from the third, fourth and fifth as well as the facial nerve are very near each other, so that what interferes with the function of one may affect the functions of one or all the others.

Dr. Henry Gradle, having observed the case, remarked first, that there was no evidence whatever of involvement of the facial nerve. Second, that as the anesthesia came on gradually, has never been complete, and as the nature and seat of the lesion is unknown, we are not justified in drawing inferences regarding the paths of the gustatory fibers. Third, the cornea was normal. He also wished to ask whether the tabetic ocular paralyses were not more complete and more fugitive than in this case. Furthermore, the impairment of sight was slight, had taken a long time to develop, and was therefore not indicative of tabes. In tabes also the loss of vision is due to visible atrophy, and this was not the case in the present observation. He could not explain the amblyopia, as there was no interference with the fields for white or colors, nor with the central perception of color. He thought it improbable that a syphilitic lesion had existed for four years, encroaching gradually from the fifth and sixth to the fourth and third nerves without causing more acute symptoms.

Dr. Hugh T. Patrick expressed the opinion that although a large proportion of the ocular paralyses of tabes are comparatively transient, and although in the permanent cases a large proportion of the patients have a high degree of paralysis with marked strabismus, yet the ocular pareses in this case were not incompatible with a diagnosis of tabes. The optic atrophy was also a somewhat anomalous condition, but there may be a stage of the disease when there is some visual defect from optic atrophy, and yet the latter be so slight that the conclusion should be drawn from the condition of vision rather than from the appearance of the disk. The atrophy might affect the central more than the other fibers, although in that case the central perception of color should be affected. He inclined to Dr. Moyer's view of the pathology of the case, that it was degenerative rather than inflammatory or neoplastic. He believed that he had seen an analogue of Moyer's first case in a patient sent to him by a laryngologist. Although a well defined anesthesia of the mucous membrane of the nose and the upper lip, where she complained of discomfort, could not be made out, it seemed to him that these branches of the fifth were involved. Examination revealed to him loss of the knee-jerk and a beginning Argyll-Robertson pupil.

Dr. Henry M. Lyman was reminded by this case of another in which there was atrophy of both optic nerves, Argyll-Robertson pupils, progressive deafness of both ears, with retention of the knee-jerks. This case seemed to be one of degenerative sclerosis involving the optic nerve and the ganglia at the base of the brain and progressing downward, the spinal cord not having yet become involved. In Dr. Brown's case the most probable hypothesis is a localized degenerative change at the point which impinges upon the roots of the nerves mentioned. Is the case tabetic in origin, or may it not be a case of progressing multiple sclerosis?

Dr. Moyer remarked in connection with the suggested diagnosis of tabes, that the bulbar forms of tabes, although exceptional, bear a strong resemblance to the ordinary forms of tabes beginning in the spinal cord. The process is on a level with the head instead of the trunk.

Dr. Brown, in closing the discussion, stated that the neurologist could fasten almost anything upon tabes, and tabes upon almost any class of symptoms. The advocates of tabes agreed that the ocular symptoms in this case were very unusual, and that the beginning unilateral sensory symptoms so strictly confined to one nerve trunk were not likely to be tabetic. He was led to exclude tabes by the rather rapid unilateral onset, by its remaining restriction, and more than all, by the involvement of the motor branch. His view of the nature of the lesion was only a suggestion. The only external growth that could account for the symptoms would be an aneurism. The objection to this hypothesis was that the pressure would not be confined exclusively to these nerve trunks, but would produce symptoms from involvement of adjoining tissues. The hypothesis of sclerosis seemed to be hardly tenable, as sclerosis in the area involved would almost certainly involve other structures. Krause, in his monograph, refers to Dr. Schmidt's cases as the most instructive of any that have been reported. In one of his cases the ganglion was completely excised, and the woman thought that she had a better sense of taste than before. In the other cases the taste was more or less lost in the anterior two-thirds of the tongue. We must at present accept the conclusion that these fibers have most diffuse paths.

UNUSUAL CASES OF MYXEDEMA.

Dr. Sydney Kuh read a paper reporting two cases of this disease. Both patients were males, the one fifty-one, the other fifty-two years old. They presented a number of the classic symptoms of myxedema such as: Atrophy of the thyroid gland, weak and low pulse, subnormal temperature, dry cold and scaly skin, and loss of sexual power. In both cases the myxedematous infiltrations were of an unusual character. In the first it was found in the cervical region only, it was crossed by a scar from an old operation and was uncommonly firm, resembling a fibromatous keloid in appearance and consistency. When the treatment was interrupted, which happened repeatedly, the swelling would promptly reappear. In the second case there was at the time of the first examination only a slight edema of the eyelids, but after a few days a typical myxedematous infiltration was found in the cervical region, which repeatedly vanished after a short existence and then, in spite of continual treatment, reappeared for a few days.

In the second case the attendant mental disturbance consisted in many delusions of a hypochondriacal nature, with pronounced indications of mental weakness. An acute mental disturbance characterized by numerous hallucinations of an unpleasant nature and great excitement was followed by a complete loss of energy, without any other signs of mental disease or weakness.

The first patient was treated with desiccated thyroids and he continues their use up to this time. He has been well for about four years. In the second case iodothyrin was used, and this seemed to have less disagreeable secondary effects than the powdered gland. He, too, continues the drug and has had no recurrence of the disease.

Dr. J. B. Herrick had had some experience with myxedema of children, but very little with this condition in adults. The deleterious effects of thyroid extract as brought out by Dr. Kuh and observed by Dr. Roos are due to contamination. The nearer the approach to pure iodothyrin, the nearer perfection is the effect of the drug. The early search for the myxedematous deposit in the supraclavicular space is of great importance. If Dr. Kuh's observations are corroborated, and if it is found there long before it is observed in the face and other parts of the body, it will prove a valuable aid in early diagnosis. The appearance and disappearance of the swelling before treatment would seem to show that the thyroid retained what might be called an intermittent function. In the case of a cretin who lives at a distance, Dr. Herrick had found that the dose could be very accurately controlled by noting the condition of the bowels. The child who had previously been badly constipated was given just enough thyroid extract to induce one or two bowel movements a day, and in this way the right dose was apparently given.

The President had noticed some unfavorable symptoms and complications following the use of Armour's thyroid preparation, but not in myxedema. In one case of climacteric insanity the pulse became very high for a couple of hours, reaching 160 or higher. The respiration was not correspondingly affected, the temperature was 100 degrees, and the patient seemed comfortable, or at least not seriously depressed. The tachycardia disappeared in about three hours. The treatment was discontinued for the time, but was taken up later on, and eventually a complete recovery from the mental symptoms was secured.

Dr. Kuh, in closing the discussion, said that he believed the best results could be obtained in myxedema by very gradually increasing the dose until the first symptoms of intoxication appeared. In the second case reported the dose was increased until the pulse became abnormally frequent, when the dose was slightly reduced. In the first case in which the tablets were used, gastrointestinal disturbance, diarrhea, gastric distress, anorexia and increased frequency of the pulse were the chief toxic effects. The dosage was thereupon reduced.

The best results cannot be obtained by small doses, but by remaining just below the toxic stage, until the symptoms disappear. Small doses should then be given every day or every third day. Unless the patients can be watched closely, they should be kept in bed while full doses are being given.

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175. PARALYSIE GÉNÉRALE INFANTILE AYANT SIMULÉ L'IDIOTIE (Infantile General Paresis Simulating Idiocy). Toulouse et Marchand (Soc. méd. des. Hôpitaux, 1899, June 23).

The recognition that a form of dementia closely resembling general paresis may occur in the young is becoming more and more generally admitted, but, according to our authors, one point has not received sufficient attention. That is, that many cases of paresis occurring in the very young are often mistaken for cases of idiocy. In a case reported the progressive dementia commenced after a period of apparently normal development. There were inequality of pupils, speech disturbances, rapid emaciation, epileptiform attacks. The autopsy findings showed cerebral atrophy, adherent meninges, proliferation of glia, and other changes held to be typical of paresis. The child's father had died of paresis, with syphilis, and the possibility of chronic meningo-encephalitis is discussed by the writers. JELLIFFE.

176. UEBER SCHÄDELOPERATIONEN BEI EPILEPSIE (Trephining for Epilepsy). Matthiolius (Deut. Zeitsch. f. Chirurgie. 52, 1899, pp. 1 and 2.

That certain cases of traumatic Jacksonian epilepsy have been much improved is well authenticated, but the results in non-traumatic cases have not been as satisfactory. Four new cases are reported here. Two for non-traumatic epilepsy, both with negative results, and two of the traumatic Jacksonian type, with one recovery.

Matthiolius also presents the statistics of 164 cases that have been under observation, of which number 89 were non-traumatic partial epilepsies, 52 generalized traumatic types, and 23 of general idiopathic epilepsy. Of this total some 24 were healed; 25 improved; 31 died; the rest remained about the same. Thus there was 29.8 per cent. of favorable results, as against 70.2 per cent. failures. JELLIFFE.